MINISTRY OF AGRICULTURE, FISHERIES AND FOOD

Survey of cadmium in food:
First Supplementary Report

The Twelfth Report of the
Steering Group on Food Surveillance
The Working Party on the Monitoring
of Foodstuffs for Heavy Metals

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Earlier reports of the Working Party for the Monitoring of Foodstuffs for Heavy Metals are as follows:

1971 Survey of mercury in food
1972 Survey of lead in food
1973 Survey of mercury in food. A Supplementary Report
1973 Survey of cadmium in food
1975 Survey of lead in food. First Supplementary Report
1981 Survey of copper and zinc in food
1982 Survey of arsenic in food
1982 Survey of lead in food. Second Supplementary Report

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INTRODUCTION

1. The Working Party on the Monitoring of Foodstuffs for Heavy Metals was set up in 1971 following reports in the United States of high concentrations of mercury in canned tuna. It has conducted and published results of surveys of foods for the presence of mercury, lead and cadmium. The results of the surveys are submitted to the Food Additives and Contaminants Committee and the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment which tender advice to Ministers.

2. The present report summarises the results of surveys on the concentrations of cadmium in food carried out since the Survey of Cadmium in Food; the Fourth Report of the Working Party in 1973[1]. The Fourth Report of the Working Party concluded that the average total weekly intake of cadmium from food and drink was not more than 250 μg per person. The report identified the need for more information on cadmium concentrations in crops grown on land treated with sewage sludge and crops grown in the vicinity of certain industrial processes. In addition, the report concluded that people who ate large amounts of certain shellfish might consistently take in above average amounts of cadmium. Details of the background to the monitoring, its objectives and organisation may be found in the Fourth Report.

RESULTS

3. The results of analyses carried out during the period 1973 to 1981 are given in Appendix I. They are mostly expressed as mg/kg of the edible portion as received; results for fresh fish are based on wet weight unless otherwise stated. Before describing the results presented in this report it should be pointed out that the limits of determination quoted for the various analyses often differ. Atomic absorption spectroscopy is generally used for the measurement of cadmium in food. Limits of determination will depend however on the method of analysis used together with the amount and nature of the sample analysed. Thus, for a particular method of analysis, it is generally possible to determine lower concentrations of cadmium in liquid foods, such as milk, than in solid foods, such as meat or vegetables. The methods of analysis customarily used by Government Laboratories for the estimation of cadmium in food were atomic absorption spectrophotometry, using flame atomisation as described previously[1, 2], or carbon furnace atomisation. Some samples however were analysed using DC plasma emission spectrophotometry directly on the sample digest.

Dietary intake of Cadmium

4. Table 1 gives the cadmium concentrations of the food groups in the continuing Total Diet Survey. The results for 1974 to 1981 are generally similar to those reported previously. The estimated mean daily intake of cadmium is less than 20 μg corresponding to a weekly intake of less than 140 μg. The apparent increase in 1980 was due to one high result in the cereals group. Many of the results of measurements made in the estimation of the average dietary intake of cadmium were at or below the limit of determination of the

analytical method. Mean cadmium concentrations in each food group were calculated in the normal fashion by assuming that results with values below the limit of determination had values equal to the limit of determination (see Table 1). Naturally such a method of calculation is likely to lead to an overestimate of the dietary intake of cadmium. It is possible to produce a lower estimate of dietary intake of cadmium by assuming that results with values less than the limit of determination have values equal to zero. On this basis the average daily intake of cadmium for 1981 is 10 μg/person, corresponding to a weekly intake of 70 μg/person. Thus for 1981 the true average daily intake of cadmium probably lies between 10 and 17 μg/person. The concentrations of cadmium in all the food groups were generally between 0·01 and 0·02 mg/kg with the exception of the cereal group where concentrations were generally between 0·02 and 0·03 mg/kg, and the beverages and milk groups where concentrations were below the limit of determination of 0·002 and 0·005 mg/kg respectively. Any changes of concentration in the food groups from year to year should not necessarily be taken to represent a real change in concentration since they are more likely to be due to sampling variation.

5. The beverages group is diluted prior to analysis by the addition of a tea infusion. To determine the concentration of cadmium in the group on the basis of material purchased and consumed, this dilution must therefore be allowed for. In the past, a multiplication factor of 5 was used to allow for an average of 400 ml water added to a 100 g group; in the revised study the 118 g group is made up to 800 g. The calculated concentration can then be multiplied by the daily consumption figure to provide an estimate of intake from this group. Of 89 samples of water associated with these Total Diet Studies, 84 contained less than or equal to 0·002 mg/l and only one contained over 0·004 mg/l of cadmium.

Individual foodstuffs

6. Table 2 gives the results for some individual foods. The elevated mean concentration of cadmium in animal kidneys of about 0·4 mg/kg for the 296 samples compared with an average of 0·33 from 12 samples in the earlier report. The mean value in liver of just over 0·1 mg/kg was about 4 times the mean value found in muscle. Products made from brown crab meat still show the highest concentration of cadmium. Values found for crab spreads and pâtés range up to over 3 mg/kg. Bottled lobster spreads and pâtés have a mean value of 0·27 mg/kg and fish spreads and pâtés a mean value of 0·10 mg/kg.

7. Most other foods contained under 0·1 mg/kg of cadmium and were often below the limits of determination. Higher concentrations occurred in dried or concentrated products and in certain other products. However, these are unlikely to be consumed in large quantities under normal conditions. Thus, pectin (sugar beet) had a mean value of 0·26 mg/kg, tea dust 0·12 mg/kg, curry powder 0·20 mg/kg, and mustard 0·11 mg/kg; wheatgerm (one sample only) contained 0·23 mg/kg cadmium, and herbs and seasonings contained mean values of 0·14 and 0·10 mg/kg respectively. The mean cadmium concentrations in both bread and flour were 0·03 mg/kg. Concentrated baby foods together with other baby foods were also generally low in cadmium and

ranged from <0·01 mg/kg to 0·20 mg/kg. The highest value was obtained for a sample of dried baby food which contained kidney. Canned baby foods contained very similar cadmium concentrations to baby foods in glass jars. Concentrations of cadmium in canned and frozen spinach were higher than in other vegetables and were 0·08 mg/kg and 0·12 mg/kg respectively. It is well known that this particular vegetable accumulates cadmium.

8. A survey of the cadmium concentration in fish landed in the United Kingdom, similar to that summarised in the Fourth Report of the Working Party, was carried out over the period 1973 to 1976. Variations in the size of the sample analysed resulted in limits of determination of 0·2, 0·02 and 0·01 mg/kg being used. Table 3 shows that almost all the samples of fish contained cadmium below the determination limit of the method used. It has been proved in careful studies elsewhere[3, 4] that the probable true concentration for cadmium in fish muscle tissues is unlikely to exceed 0·005 mg/kg.

9. Table 4 shows the concentrations of cadmium found in freshwater species collected from lochs, rivers and fish farms in Scotland. Although the food of the various species varies widely the cadmium concentrations are not raised, nor are there significant differences in the metal concentrations in fish taken from fish farms or the wild.

10. The results for shellfish taken from coastal waters of the United Kingdom are summarised in Table 5. Where direct comparisons are possible with similar results in the Fourth report of the Working Party, there is general agreement. Brown crab meat contains the highest cadmium concentration, ranging from <0·2 up to 86 mg/kg. As with the earlier report the concentration of cadmium in claw or white crab meat is considerably lower, most values being below the limit of determination of 0·2 mg/kg, with an upper limit of 2·9 mg/kg. Lobster brown meat had an average cadmium concentration of 1·8 mg/kg whilst that in the claw and tail meat was in all cases below 0·2 mg/kg. The average concentration of cadmium in whelks of 1·1 mg/kg compares with a value of 0·7 mg/kg found in the first survey. Oysters contained just under 0·4 mg/kg with a maximum of 1·8 mg/kg of cadmium and scallops (white meat) had an average concentration of 0·5 mg/kg. Shrimps, winkles, mussels and cockles generally contained less than 0·2 mg/kg cadmium, whilst queens (muscle) had an average concentration of cadmium of 0·5 mg/kg. As in the previous survey there were considerable regional variations in those species containing elevated cadmium concentrations. The highest average concentrations of cadmium in crabs were recorded off the south-west coast of England and the Welsh coast. There was a tenfold difference in the concentrations in brown crab meat from this area compared with those from the east coast of England.

11. Duplicate diets from high fish consumers from the north-east Irish Sea coastal areas as well as from fishing communities in the south-west of England which were collected for a study designed to estimate their mercury intakes were also analysed for cadmium. The results are given in Table 6. Neither of

these groups could be strictly considered as a 'critical group'* as far as cadmium was concerned. The average consumption of fish and shellfish in the two groups was 60·3 g/day, three times the national average. The average cadmium intake for the 174 individuals in the survey was 0·12 mg/week from whole diets which compares with a figure of 0·126 mg/week calculated from Total Diet samples also collected in 1977. The diets collected from the fishing communities, however, weighed less than those collected for the Total Diet Study. Nine individuals (5·2 per cent) had cadmium intakes greater than 0·4 mg/week. Although the fish consumption of this group, at 60·7 g/day, was similar to that of the whole study population, the fish portion of the diet contributed the major part of these high cadmium intakes. Consumption of shellfish was significantly associated ($P < 0{\cdot}001$) with the highest cadmium intakes in this study.

12. Subsequent to these duplicate diet studies further studies were made with the co-operation of high fish consumers living in the Thames Estuary area and in areas in North Wales and north-west England. These people provided duplicates of the fish and shellfish component of their diets eaten during one week and kept a diary record of their consumption of fish and shellfish for one month, including the week of the duplicate diet. Almost all those people whose duplicate diets contained cadmium at concentrations greater than the limit of determination had eaten shellfish. The weekly cadmium intake **from fish** of the people who did not eat shellfish was 3 μg/person. In contrast the mean weekly cadmium intake from fish and shellfish of the people consuming both was 40 μg/person. These data provide reasonable evidence to show that elevated cadmium intakes are associated with people consuming shellfish. This supports the tentative conclusion reached by the Working Party when it first reported on cadmium. Other studies on shellfish consumption are in progress; these studies are designed to provide information on the likely cadmium intake of people who regularly consume shellfish.

13. Table 7 summarises the results of a survey over the period 1976 to 1977 of the cadmium concentrations in vegetables grown on land known not to have been treated with sewage sludge. Samples of produce were obtained at various establishments from plots whose history was known with some confidence. All mean cadmium concentrations were below 0·1 mg/kg on a wet weight basis. Although samples of carrots, parsnips, leeks, lettuce and oats occasionally exceeded this value, only seven out of a total of 334 contained more than 0·1 mg/kg and of these, three were lettuce samples.

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*Critical groups comprise people whose exposure to a particular contaminant is unusually high. This may arise either (a) because they have atypical food consumption patterns or (b) because their metabolism is atypical (e.g. children) or (c) because they are exposed to an above average concentration of the particular contaminant present in their diet. Typically, cases of unusual exposure to cadmium may arise in groups of people who consume large quantities of certain shellfish or shellfish products or who consume food grown on contaminated land.

AREAS OF POTENTIAL CADMIUM CONTAMINATION

Sewage sludge

14. Tables 8 and 9 show the results of analyses of vegetables grown in the Heathrow area where continued use of sewage sludge over a long period has led to raised cadmium concentrations in the soil. It is clear that increased soil cadmium concentrations lead to raised cadmium concentrations in vegetables grown in such soil. This increase is most marked in lettuce, with a maximum concentration of 1·79 mg/kg, parsnips with a maximum of 1·47 mg/kg, leeks with a maximum of 0·71 mg/kg and carrots with a maximum of 0·69 mg/kg of cadmium. All figures are on a fresh weight basis. Soil cadmium concentrations ranged from 0·5 to 34 mg/kg. A duplicate diet study was carried out in 1978 on 21 people eating vegetables from these sites and their total week's diet, excluding milk and beverages, was analysed. Cadmium concentrations in the diets were found to be higher than the national average. A mean value of 23 μg/kg was obtained compared with a mean value of 17 μg/kg from the Total Diet Study. Results from a four-week diary study carried out in 1979 on the same group coupled with the results from crop analyses confirm that cadmium concentrations in the diets of this population are above average. Actual intakes however, are unlikely to exceed the FAO/WHO provisional tolerable weekly intake of 400 – 500 μg cadmium.

Shipham

15. High cadmium concentrations have been detected in the soil at Shipham, a village in Somerset. Part of the village was built on the spoil heaps arising from zinc mining activities. Extensive follow-up studies have now been performed in the village under the auspices of the Local Authority, MAFF and DoE. Table 10 shows the results of a survey of cadmium concentrations in fruit and vegetables grown in gardens and allotments in the area. Average soil cadmium concentrations ranged from 2 – 520 mg/kg dry soil. For vegetables, mean concentrations of cadmium were highest in herbs, celery (1·0 mg/kg) and leafy vegetables, such as spinach (0·79 mg/kg), kale (0·75 mg/kg), lettuce (0·68 mg/kg) and cabbage (0·61 mg/kg). Of the fruits examined, rhubarb showed the highest cadmium concentrations, giving a mean concentration of 0·42 mg/kg. A comparison of the results for cadmium concentrations in vegetables grown at Shipham, Heathrow and in non-contaminated soils is given in Table 11. This shows that the concentrations of cadmium in vegetables from Shipham are generally three to four times those found in vegetables grown in soil near Heathrow which was heavily contaminated with cadmium by the continued use of sewage sludge. The cadmium concentrations in vegetables from both Shipham and Heathrow are up to a factor of 10 or more higher than the concentrations found in vegetables grown in uncontaminated soil. Wide variations in cadmium uptake were found for most of the crops studied. All analyses were carried out on fruit and vegetables prepared as for cooking or for direct consumption. A small amount of data is also available which suggests that further losses of cadmium occur during the cooking process.

16. The extent of cadmium uptake from soil into crops is known to be dependent on various factors such as soil cadmium concentration, soil pH,

rate of growth of crop and even the cultivar of crop. Little data on cultivar variation is available and most of the work has been carried out on lettuces. Real differences have been shown to exist between cultivars in their ability to take up cadmium via the roots and translocate it to the lettuce leaves. The differences do not appear to be related to the type of lettuce, e.g. whether butterhead, crisp or cos; there is, however, little doubt that they are genetically controlled but by genes different from those which control the form of plant produced.

17. Results of a one-week duplicate diet study (65 individuals) and two four-week diary studies (80 – 85 households) at Shipham have shown that although the cadmium concentrations are on average nearly double those found in the national average diet, only a very small percentage of the Shipham population studied (4 per cent) is likely consistently to exceed the intake of 400 – 500 μg cadmium, currently considered tolerable by the Joint United Nations Food and Agriculture Organisation/World Health Organisation Joint Expert Committee on Food Additives (JECFA). The latter intake is known as the Provisional Tolerable Weekly Intake and includes a safety factor. On the basis of medical checks completed in the village to date there is no evidence of any current health problems directly related to exposure to cadmium.

Cadmium pigments

18. Cadmium may also be present in food as a result of its use in food contact materials and articles. Cadmium pigments are used to decorate such articles of ceramic ware and vitreous enamel-ware; however, in each case, permissible levels of release of cadmium are strictly controlled by legislation[5,6]. Cadmium pigments are also used to colour certain plastics, although extensive laboratory studies in Germany have demonstrated that the release of cadmium from such plastics is very small[7,8]. In the United Kingdom investigations have indicated that the usage of plastics coloured with cadmium pigments for purposes which will bring them into direct contact with food is minimal. Cadmium-containing compounds may be employed as stabilisers in polyvinyl chloride materials and articles, but once again enquiries have revealed no significant usage of these for food contact purposes. Therefore it may be concluded that any intake of cadmium from food contact materials and articles is likely to be minute.

APPRAISAL OF RESULTS

19. From the results of the analyses of the Total Diet samples it is estimated that the intake of cadmium from the 1·46 kg of food consumed daily by the average person in the United Kingdom is under 20 μg, which is equivalent to a weekly intake of not more than 140 μg. This is well within the provisional tolerable weekly intake of cadmium of 400 – 500 μg per adult proposed by the Joint FAO/WHO Expert Committee on Food Additives[9]. In general, neither water nor air is likely to be a significant source of cadmium. Tap water usually contains under 1 μg/l of cadmium[10, 11, 12] although, if galvanised pipes are used in the supply, concentrations above this may arise. The water sam-

ples obtained in the Total Diet Studies (paragraph 5) were mostly below 1 μg/l which was usually the limit of determination. Average (annual) concentrations in air rarely exceed 0·02 μg/m^3 even in urban areas[13]. Even at this concentration, intakes are unlikely to exceed 2 μg/week and 70 – 80 per cent of this is likely to be exhaled immediately.

20. Daily intakes of cadmium estimated for the USA, Romania, Czechoslovakia and non polluted areas of Japan were quoted in the first MAFF Report of Cadmium in Food[1]. Estimates have been published since then from many countries and using a variety of techniques; some are reviewed by the Commission of the European Communities who concluded[14] that intakes from food averaged 43 μg/day (range 4 – 84 μg/day). Estimates of average cadmium intake, excluding those relating to children, published since then range from 17 μg/day in Australia[15] to 59 μg/day in control areas in Japan, and West Germany[16, 17]. Differences in analytical methodology and in the methods used for estimating the amount of food ingested per day probably contributed to the wide variation obtained. The current estimate of a daily intake of about 20 μg in the United Kingdom comes within the range of 4 to 80 μg of the above national estimates. Calculation of the intake of cadmium using the upper limit of the ranges of cadmium concentration in Table 1 for 1981 gives an upper limit for the intake from food of about 24 μg per day.

Individual foods

21. Cadmium is present in most foods in the United Kingdom at low concentrations. Very few foods have cadmium concentrations above 1 mg/kg and most mean values are an order of magnitude lower. Of the foods mentioned in paragraphs 6 and 7 which show cadmium concentration higher than the majority, those prepared from brown crab meat contain the highest concentration of cadmium. Bottled crab pastes have an average cadmium concentration of 0·52 mg/kg and an upper value of 1·5 mg/kg. Bottled and canned crab pâtés and spreads have an average cadmium concentration of 1·9 mg/kg and an upper value of 3·2 mg/kg.

Areas of potential Cadmium contamination

22. Tables 7 to 11 show very clearly that raised concentrations of cadmium in soils lead to elevated concentrations of the element in vegetables and fruit grown on the soils in agreement with many reports in the literature. Dietary studies of people eating vegetables from market gardens near Heathrow show that they have an elevated cadmium intake. However, as reported in Paragraph 14, the market gardeners and their families did not have cadmium intakes in excess of the PTWI. In contrast, studies at Shipham have shown that the average concentration of cadmium in the diets of participants was considerably higher than the national average concentration and that a small proportion of participants exceeded the PTWI. Intakes of cadmium at Shipham could be reduced by villagers consuming less home-grown produce, as they have been advised to do on several occasions.

CONCLUSIONS

23. (i) It is estimated from the Total Diet Study that the intake of cadmium from the 1·46 kg of food consumed daily by the average person in the United Kingdom is under 20 μg. The average total weekly intake from food and drink amounts to not more than 140 μg.

(ii) Cadmium occurs at very low concentrations in the general food supply including fish.

(iii) The main sources of high cadmium concentrations in the general food supply are kidney and brown crab meat. People who regularly consume kidney and brown crab meat will undoubtedly have elevated cadmium intakes. The Working Party have initiated a programme of research to assess the extent of absorption of cadmium in human volunteers from crab meat.

(iv) Vegetables grown in soil with elevated concentrations of cadmium arising from past metal workings or long-term applications of sewage sludge themselves contain high concentrations of cadmium. Research efforts to establish the distribution and intensity of this problem should be stepped up with the aim of limiting the effect upon cadmium intakes from food.

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APPENDIX I

RESULTS OF ANALYSES

List of Tables

1. Cadmium in total diet samples and daily intake of cadmium (1974 – 1981).
2. Cadmium in individual food items of the national diet.
3. Cadmium in fish caught in UK coastal and distant waters (1973 – 1976).
4. Cadmium in freshwater fish from Scotland.
5. Cadmium in shellfish landed in the United Kingdom (1973 – 1976).
6. Cadmium intakes of high fish consuming groups.
7. Survey of Cadmium in vegetables grown on land not treated with sewage sludge (1976 – 1977).
8. Cadmium in vegetables grown on experimental plots near to Heathrow.
9. Cadmium in vegetables grown in Heathrow market gardens (1978 – 1979).
10. Cadmium in fruit and vegetables grown at Shipham.
11. Comparison between cadmium in vegetables from Shipham and from Heathrow with cadmium in vegetables grown in non-contaminated soils.

All the results for analyses in these tables are reported in the form of mg/kg fresh weight unless otherwise stated.

APPENDIX I

Table 1: Cadmium in total diet samples and daily intake of cadmium 1974 – 1981(a)

Food group	Estimated weight of food eaten (kg/day)	Number of samples	Range (mg/kg)	Mean value (mg/kg)	Estimated mean daily intake (μg/person)
1974					
1. Cereals	0·27	26	<0·01–0·08	<0·020	<5
2. Meat and fish	0·18	26	<0·01–0·09	<0·021	<4
3. Fats	0·08	26	<0·01–0·07	<0·016	<1
4. Fruit and preserves	0·25	26	<0·01–0·02	<0·013	<3
5. Root vegetables	0·21	26	<0·01–0·06	<0·019	<4
6. Green vegetables	0·11	26	<0·01–0·03	<0·012	<1
7. Milk	0·40	26	<0·005–0·01	<0·006	<2
Total	1·50				<20
8. Fish		26	<0·01–0·02	<0·012	
1975 (first three quarters)					
1. Cereals	0·24	13	0·01–0·04	0·022	5
2. Meat and fish	0·19	13	<0·01–0·11	<0·032	<6
3. Fats	0·08	13	<0·01–0·02	<0·012	<1
4. Fruit and preserves	0·19	13	<0·01–0·02	<0·011	<2
5. Root vegetables	0·22	13	<0·01–0·09	<0·019	<4
6. Green vegetables	0·13	13	<0·01–0·03	<0·012	<2
7. Milk	0·40	13	<0·005	<0·005	<2
Total	1·47				<22
8. Fish		13	<0·01–0·01	<0·01	
1975 (fourth quarter)					
1. Cereals	0·23	8	0·02–0·03	0·023	5
2. Meat	0·15	8	<0·01–0·04	<0·016	<2
3. Fish	0·02	8	<0·01–0·03	<0·019	<0·4
4. Fats	0·08	8	<0·01–0·01	<0·010	<1
5. Fruit and preserves	0·17	8	<0·01–0·02	<0·011	<2
6. Root vegetables	0·18	8	<0·01–0·03	<0·019	<3
7. Other vegetables	0·11	8	<0·01–0·03	<0·015	<2
8. Beverages	0·12	8	<0·005	<0·005	<3(b)
9. Milk	0·40	8	<0·005	<0·005	<2
Total	1·46				<20

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Table 1 (Cont'd)

Food group	Estimated weight of food eaten (kg/day)	Number of samples	Range (mg/kg)	Mean value (mg/kg)	Estimated mean daily intake (μg/person)
1976					
1. Cereals	0·23	25	<0·01–0·04	<0·021	<5
2. Meat	0·15	25	<0·01–0·08	<0·018	<3
3. Fish	0·02	25	<0·01–0·02	<0·013	<0·3
4. Fats	0·08	25	<0·01–0·01	<0·010	<1
5. Fruits and sugars	0·17	25	<0·01–0·03	<0·011	<2
6. Root vegetables	0·18	25	<0·01–0·05	<0·018	<3
7. Other vegetables	0·11	25	<0·01–0·03	<0·012	<1
8. Beverages	0·12	25	<0·005	<0·005	<3(b)
9. Milk	0·40	25	<0·005–0·01	<0·005	<2
Total	1·46				<20
1977					
1. Cereals	0·23	25	0·01–0·04	0·022	5
2. Meat	0·15	25	<0·01–0·07	<0·020	<3
3. Fish	0·02	25	<0·01–0·05	<0·015	<0·3
4. Fats	0·08	25	<0·01–0·03	<0·012	<1
5. Fruits and sugars	0·17	25	<0·01–0·03	<0·012	<2
6. Root vegetables	0·18	25	<0·01–0·04	<0·018	<3
7. Other vegetables	0·11	25	<0·01–0·02	<0·011	<1
8. Beverages	0·12	25	<0·002–0·004	<0·002	<1(b)
9. Milk	0·40	25	<0·005–0·005	<0·005	<2
Total	1·46				<18
1978					
1. Cereals	0·23	20	0·01–0·04	0·029	7
2. Meat	0·15	20	<0·01–0·02	<0·011	<2
3. Fish	0·02	20	<0·01–0·32	<0·037	<1
4. Fats	0·08	20	<0·01–0·02	<0·011	<1
4. Fruits and sugars	0·17	20	<0·01–0·03	<0·011	<2
6. Root vegetables	0·18	20	<0·01–0·04	<0·017	<3
7. Other vegetables	0·11	20	<0·01–0·02	<0·012	<1
8. Beverages	0·12	20	<0·002–0·006	<0·002	<1(b)
9. Milk	0·40	20	<0·005–0·005	<0·005	<2
Total	1·46				<20

Table 1 (Cont'd)

Food group	Estimated weight of food eaten (kg/day)	Number of samples	Range (mg/kg)	Mean value (mg/kg)	Estimated mean daily intake (μg/person)
1979					
1. Cereals	0·23	19	0·01–0·04	0·020	5
2. Meat	0·15	19	<0·01–0·04	<0·013	<2
3. Fish	0·02	19	<0·01–0·02	<0·012	<0·2
4. Fats	0·08	19	<0·01–0·01	<0·010	<1
5. Fruits and sugars	0·17	19	<0·01–0·01	<0·010	<2
6. Root vegetables	0·18	19	<0·01–0·04	<0·017	<3
7. Other vegetables	0·11	19	<0·01–0·02	<0·012	<1
8. Beverages	0·12	19	<0·002–0·004	<0·002	<1(b)
9. Milk	0·40	19	<0·005	<0·005	<2
Total	1·46				<17
1980					
1. Cereals	0·23	16	0·02–0·41	0·050	12
2. Meat	0·15	16	<0·01–0·02	<0·012	<2
3. Fish	0·02	16	<0·01–0·03	<0·013	<0·3
4. Fats	0·08	16	<0·01–0·01	<0·010	<1
5. Fruits and sugars	0·17	16	<0·01–0·01	<0·010	<2
6. Root vegetables	0·18	16	<0·01–0·03	<0·015	<3
7. Other vegetables	0·11	16	<0·01–0·03	<0·014	<2
8. Beverages	0·12	16	<0·002–0·022	<0·003	<2(b)
9. Milk	0·40	16	<0·005–0·005	<0·005	<2
Total	1·46				<26
1981					
1. Cereals	0·23	8	0·02–0·03	0·021	5
2. Meat	0·15	8	<0·01–0·02	<0·011	<2
3. Fish	0·02	8	0·01–0·02	0·013	0·3
4. Fats	0·08	8	<0·01–0·01	<0·010	<1
5. Fruits and sugars	0·17	8	<0·01–0·01	<0·010	<2
6. Root vegetables	0·18	8	<0·01–0·03	<0·015	<3
7. Other vegetables	0·11	8	<0·01–0·02	<0·011	<1
8. Beverages	0·12	8	<0·002–0·002	<0·002	<1(b)
9. Milk	0·40	8	<0·005	<0·005	<2
Total	1·46				<17

Table 1 (Cont'd)

Food group	Estimated weight of food eaten (kg/day)	Number of samples	Range (mg/kg)	Mean value (mg/kg)	Estimated mean daily intake (μg/person)
1981 Revised Study					
1. Bread and cereals	0·240	10	0·02–0·03	0·021	5
2. Meat and poultry	0·058	10	<0·01–0·01	<0·010	<1
3. Offal	0·003	10	0·05–0·16	0·089	0·3
4. Meat products	0·077	10	<0·01–0·01	<0·010	<1
5. Fish	0·015	10	<0·01–0·06	<0·015	<0·2
6. Oils	0·090	10	<0·01–0·01	<0·010	<1
7. Sugar and preserves	0·095	10	<0·01–0·01	<0·010	<1
8. Green vegetables	0·046	10	<0·01–0·01	<0·010	<0·5
9. Potatoes	0·159	10	<0·01–0·02	<0·013	<2
10. Other vegetables	0·068	10	<0·01–0·01	<0·010	<1
11. Canned vegetables	0·043	10	<0·01–0·02	<0·011	<0·5
12. Fresh fruit	0·060	10	<0·01–0·01	<0·010	<1
13. Fruit products	0·024	10	<0·01–0·01	<0·010	<0·2
14. Beverages	0·118	10	<0·002	<0·002	<2(b)
15. Milk	0·360	10	<0·005	<0·005	<2
Total	1·456				<19

Means have been calculated assuming that all figures quoted as less than the limit of determination are equal to the limit.

(a) Results obtained by the Laboratory of the Government Chemist.

(b) Corrected for dilution as indicated in paragraph 5.

Table 2: Cadmium in individual food items of the national diet[a]

Food	Number of samples	Mean (mg/kg)	Range (mg/kg)
Meat and Fats			
Calf—kidney	27	0·20	0·01–0·70
Beef—liver (ox)	4	0·12	0·09–0·21
—kidney	44	0·48	0·04–2·7
—other meat	72	0·03	0·002–0·18
Pork—liver	4	0·13	0·07–0·23
—kidney	192	0·45	0·05–2·60
—other meat	92	0·03	0·006–0·18
Lamb—liver	4	0·05	0·02–0·08
—kidney	33	0·17	0·02–0·94
—other meat	69	<0·02	<0·002–0·09
Poultry	28	<0·05	<0·01–0·27
Butter	5	0·02	0·01–0·03
Cheese	5	0·03	0·03–0·05
Cooking Oils	5	0·03	0·03–0·04
Eggs	5	<0·03	<0·03–0·04
Lard	2	0·02	0·02–0·03
Margarine	3	<0·02	<0·01–0·04
Cereals, Dried and Concentrated Foods			
Agar agar	1	0·06	
Baby foods (dry) without offal	14	<0·03	<0·01–0·13
Baby foods (dry) with offal	1	0·20	
Baking powder	1	<0·01	
Bread—white	10	0·02	0·02–0·03
—wholemeal	10	0·03	0·02–0·03
Caramel	1	0·02	
Coffee—beans	3	0·04	0·02–0·06
—instant	17	<0·03	<0·01–0·15
—essence (with chicory)	1	0·01	
Cream of Tartar	1	<0·01	
Curry powder	4	0·20	0·02–0·30
Dehydrated vegetables	18	<0·11	<0·01–0·32
Dextrose	1	0·02	
Dried milk	5	<0·02	<0·01–0·03
Evaporated and condensed milk	5	<0·01	<0·01–0·01
Flour—plain	5	0·03	0·02–0·04
—self raising	5	0·03	0·03–0·04
—wholemeal	5	0·03	0·03–0·05
Food dyes and flavours	14	<0·01	<0·01–0·03
Fruit juices (frozen concentrated)	3	<0·01	
Gelatine	9	<0·02	<0·01–0·08
Glucose	2	<0·01	
Gravy mixes	5	<0·01	<0·01–0·02
Herbs	5	0·14	0·06–0·25
Jellies	5	<0·01	<0·01–0·01
Lecithin	2	0·01	
Meat extracts	4	<0·02	<0·01–0·03
Milk drink	1	0·02	

Table 2: (Cont'd)

Food	Number of samples	Mean (mg/kg)	Range (mg/kg)
Cereals, Dried and Concentrated Foods (Cont'd)			
Mustard	8	0·11	0·03–0·34
Pectin—sugar beet	2	0·26	0·19–0·33
Pectin—citrus	6	0·02	0·01–0·03
Salt	15	<0·005	
Seasonings	4	0·10	0·02–0·17
Seaweed extract	2	0·05	0·02–0·08
Soft drinks—concentrated	4	<0·01	
Soup powders	3	<0·01	
Spices	10	<0·08	<0·01–0·20
Stock cubes—beef	4	<0·01	<0·01–0·02
—chicken	4	<0·03	<0·01–0·04
Tea—leaves	17	<0·03	<0·01–0·08
—dust	14	<0·12	<0·01–0·90
—instant	2	0·01	
Tomato puree (tube)	3	0·06	0·05–0·06
Wheat gluten	2	0·02	
Wheat germ	1	0·23	
Yeast	3	0·08	0·07–0·08
Sugar and Confectionery			
Raw sugar	4	<0·02	<0·01–0·03
Sugar—white	4	<0·02	<0·01–0·02
—brown	4	<0·01	<0·01–0·02
—icing	2	0·01	
Chocolate confectionery	5	0·02	0·01–0·04
Sugar confectionery	5	<0·01	<0·01–0·01
Bottled Foods			
Baby foods without offal	9	<0·01	<0·01–0·01
Baby foods with offal	6	<0·03	<0·01–0·05
Beers and ciders	5	<0·01	<0·01–0·03
Chutneys	2	<0·01	
Crab pastes	6	0·52	0·09–1·5
Crab spreads and pâtés	8	1·88	0·03–3·2
Cream	2	0·03	0·02–0·04
Fish pastes	13	<0·04	<0·01–0·13
Fish spreads and pâtés	14	<0·10	<0·01–1·0
Fruit juices	17	<0·01	<0·01–0·02
Lobster spreads and pâtés	3	0·27	0·04–0·41
Meat paste	2	<0·02	<0·01–0·02
Milk	154	<0·002	<0·001–0·006
Soft drinks	5	<0·01	<0·01–0·01
Spirits	5	<0·01	<0·01–0·01
Wine—white	3	<0·01	
—red	2	<0·01	
Canned Foods			
Baby foods without offal	8	<0·01	<0·01–0·02
Baby foods with offal	7	0·02	0·01–0·05
Beer	4	<0·001	

Table 2 (Cont'd)

Food	Number of samples	Mean (mg/kg)	Range (mg/kg)
Canned Foods (Cont'd)			
Crab spreads and pâtés	2	1·95	1·9–2·0
Cream	2	<0·01	
Fish paste	1	<0·02	
Fruit			
Apples	2	<0·01	<0·01–0·01
Apricots	2	<0·01	<0·01–0·01
Blackberries	2	<0·01	<0·01–0·01
Blackcurrants	2	<0·01	
Cherries (fruit)	4	<0·03	<0·01–0·08
(juice)	4	<0·01	
Grapefruit (fruit)	2	<0·01	
(juice)	2	<0·03	<0·01–0·04
Mandarin oranges	2	<0·01	
Peaches	2	<0·01	
Pineapple	2	<0·01	
Plums (fruit)	3	<0·04	<0·01–0·09
(juice)	3	<0·01	<0·01–0·02
Raspberries (fruit)	2	<0·02	<0·01–0·02
(juice)	2	<0·01	
Other fruit	8	<0·01	
Fruit juice	50	<0·01	<0·01–0·02
Lobster spreads and pâtés	2	0·05	0·04–0·05
Meat	10	<0·04	<0·01–0·12
Sardines	5	0·01	0·01–0·02
Soft drinks	5	<0·001	<0·001–0·001
Tomato purée	3	0·05	0·03–0·07
Vegetables			
Beans—baked	4	<0·01	<0·01–0·02
—broad	1	<0·01	
—green	1	<0·01	
—haricot	1	<0·01	
Carrots	3	<0·01	<0·01–0·02
Mushrooms	2	<0·01	
Peas	3	<0·01	
Spinach	5	0·08	0·02–0·13
Other vegetables	5	<0·01	
Vegetable cocktail juice	1	0·01	
Frozen Foods			
Broad beans	3	<0·02	
Broccoli	2	0·01	
Brussels Sprouts	3	<0·01	
Fish Fingers (breaded)	3	<0·05	<0·04–0·06
Spinach	3	0·12	0·05–0·19
Sweetcorn	3	<0·03	<0·02–0·05
Shellfish (from the Thames Estuary)			
Cockles	1	0·14	
Cockles (cooked)	1	0·12	
Mussels	3	0·94	0·51–1·6

Table 2 (Cont'd)

Food	Number of samples	Mean (mg/kg)	Range (mg/kg)
Shellfish (from the Thames Estuary) (Cont'd)			
Whelks	6	1·03	0·50–2·20
Whelks (cooked)	1	1·30	

(a) Results provided by Department of Agriculture for Northern Ireland, Laboratory of the Government Chemist, Agricultural Service Laboratory, Food Science Laboratory.

Table 3: Cadmium in fish caught in UK coastal and distant waters (1973–1976) (mg/kg fresh weight)(a)

Species	Total no. of samples	Mean(b)	Range	No. recorded(c) <0·2	No. recorded <0·02
Bass	14	<0·2	<0·2– 0·2	13	
Bib	52	<0·15	<0·02–<0·3	25	19
Catfish	10	<0·2		10	
Cod	343	<0·16	<0·02– 0·4	169	70
Coley	350	<0·03	<0·01–<0·4		170(d)
Dab	146	<0·2	<0·02–<0·4	79	10
Dogfish	89	<0·18	<0·02–<0·2	79	10
Dover sole	16	<0·2		16	
Flounder	20	<0·3	<0·3–<0·4		
Grenadier	15	<0·2		15	
Gurnard	45	<0·2		45	
Haddock	111	<0·09	<0·02–<0·2	41	70
Hake	55	<0·17	<0·02– 0·6	24	10
Halibut	46	<0·06	<0·02–<0·2	10	36
Herring	171	<0·18	<0·02– 0·4	141	20
Horse mackerel	30	<0·2		30	
John Dory	1	<0·4			
Lemon sole	44	<0·16	<0·02–<0·2	34	10
Ling	35	<0·16	<0·02–<0·4	23	10
Mackerel	176	<0·13	<0·02–<0·3	66	60
Megrim	10	<0·02			10
Monkfish	10	<0·02			10
Plaice	370	<0·17	<0·02–<0·4	115	70
Pollack	10	<0·2		10	
Poor	8	<0·2		8	
Rabbitfish	9	<0·2		9	
Ray	57	<0·2		57	
Redfish	10	<0·2		10	
Scabbard	24	<0·2		24	
Scad	3	<0·2		3	
Skate	10	<0·02			10
Sole	50	<0·24	<0·2–<0·3	30	
Sprat	40	<0·16	<0·02–<0·2	30	10
Spurdog	10	<0·2		10	
Whiting	360	<0·17	<0·02– 0·2	240	70
Blue whiting	54	<0·2	<0·2– 0·7	34	
Witch	31	<0·09	<0·02– 0·3	10	20
Total	2805			1400	695

(a) Results of surveys carried out by MAFF Fisheries Laboratories and DAFS Marine Laboratories.

(b) Following usual practice, in calculating means, values below the limit of determination are taken to be at that value. However, as so many samples contained cadmium at levels below the limit of determination, the number of samples with these levels are given.

(c) Excludes those separately recorded as <0·02.

(d) Includes 100 samples recorded as <0·01.

Table 4: Cadmium in freshwater fish from Scotland (mg/kg fresh weight)(a)

Species	No. of samples	Mean	Range
Brown Trout	67	<0·025	<0·005–0·26
Rainbow Trout	51	<0·018	<0·005–0·07
Sea Trout(b)	68	<0·029	<0·01–0·07
Eels	7	0·09	0·05–0·12
Grayling	8	0·041	0·01–0·13
Pike	16	0·036	0·03–0·05
Atlantic Salmon(b)	17	all <0·03	

(a) Results of surveys by DAFS Laboratories.

(b) Though considered a freshwater fish, the period of maximum growth and feeding of this species is during their time at sea. The samples of sea trout were collected from coastal waters, and the Atlantic salmon were collected from freshwater.

APPENDIX I

Table 5: Cadmium in shellfish landed in the United Kingdom (1973–1976)(a) (mg/kg fresh weight)

Source	Shellfish other than crabs					Crabs					
	Number of analyses	Number of samples	Species	Range	Mean	Number of analyses	Number of samples	Brown Meat		White Meat	
								Range	Mean	Range	Mean
Orkney to Shetland	9	9	Lobster (claw)	<0·02–0·30	<0·07	28	28	0·63–62·5	11·2	<0·02–0·44	<0·07
	10	10	Lobster (tail)	<0·02–0·06	<0·03						
	16	16	Scallop (whitemeat)	0·19–0·84	0·44			Processed			
	14	14	Scallop (gonad)	0·39–1·54	0·73	121	121(b)	0·6–49·2	8·9	0·08–0·48	0·23(c)
Berwick to Sunderland	4	6	Whelks	<0·2–3·0	<1·8	44	44	<0·2–12·4	4·3(d)		
	7	223	Pink shrimps		<0·2					<0·2–0·3	<0·2
	30	30	Scampi (tail)		<0·2						
	4	240	Brown shrimps	<0·2–0·4	<0·3						
	1	250	Winkles		<0·2						
	2	124	Mussels	<0·2–0·4	<0·2						
Sunderland to Whitby	5	101	Scampi (tail)		<0·2	77	77	0·30–50·0	2·8	<0·1–0·23	<0·1
	10	16	Whelks	<0·2–5·0	<1·4						
	3	160	Pink shrimps		<0·2						
	2	82	Brown shrimps		<0·2						
	36	36	Lobster (body)	<0·2–1·60	<0·81						
	36	36	Lobster (tail)		<0·2						
	30	30	Lobster (claw)		<0·2						
Whitby to Mablethorpe	10	28	Whelks	<0·2–2·0	<0·7	74	74	0·3–17·2	3·2	<0·1–0·63	<0·16(e)
	1	180	Pink shrimps		<0·2						

Table 5 (Cont'd)

Source	Shellfish other than crabs					Crabs					
	Number of analyses	Number of samples	Species	Range	Mean	Number of analyses	Number of samples	Brown Meat		White Meat	
								Range	Mean	Range	Mean
Mablethorpe to Hunstanton	1	1	Whelks		1·6						
	1	120	Pink shrimps		<0·2						
	2	80	Portuguese oysters	0·4–0·5	0·4						
	3	256	Cockles		<0·2						
	1	50	Native oysters		0·5						
	1	19	Japanese oysters		0·3						
	2	78	Mussels	0·2–0·3	0·25						
Hunstanton to Harwich	3	41	Whelks	0·3–1·9	1·2	66	66	0·4–16·0	4·8	0·1–0·3	<0·15
	4	201	Brown shrimps	<0·2–1·3	<0·5						
Harwich to Margate	1	1	Whelks		2·5						
	1	178	Brown shrimps		<0·2						
	1	236	Pink shrimps		<0·2						
	2	330	Winkles		<0·2						
	1	124	Cockles		<0·2						
	3	120	Native oysters	0·4–1·2	0·7						
	7	350	Portuguese oysters	0·3–1·2	0·8						
Margate to Beachy Head	3	42	Whelks	0·2–5·3	1·8						
Beachy Head to Portland Bill	6	6	Oysters	0·3–0·5	0·4	158	148	0·07–33·0	4·5	0·01–1·5	0·13(f)
	1	17	Whelks		0·3						
	1	240	Winkles		<0·2						
	1	93	Cockles		<0·2						
	2	65	Portuguese oysters		0·2						
	4	132	Native oysters	<0·2–0·4	<0·3						
	3	31	Clams		<0·2						

Table 5 (Cont'd)

Source	Shellfish other than crabs					Crabs					
	Number of analyses	Number of samples	Species	Range	Mean	Number of analyses	Number of samples	Brown Meat		White Meat	
								Range	Mean	Range	Mean
Portland Bill to St Austell Bay	3	139	Mussels		<0·2	115	115	1·1–72·0	19·5	<0·1–2·9	<0·16(g)
	6	228	Queens (muscle)	<0·2–0·9	<0·5						
	3	143	Queens (gonad)	0·3–1·1	0·73						
	5	123	Scallops (muscle)	0·40–0·8	0·5						
	4	99	Scallops (gonad)	0·20–0·8	0·5						
	1	142	Winkles		<0·2						
	1	74	Cockles		<0·2						
	3	106	Portuguese oysters	<0·2–0·3	<0·2						
	1	50	Native oysters		0·3						
St Austell Bay to Hartland Point						70	70	<0·2–49·0	9·5	<0·1–0·5	<0·2
Hartland Point to St David's Head	1	25	Mussels		0·4	30	30	5·0–52·0	22·1	<0·1–0·7	<0·17
	1	12	Oysters		1·5						
	9	9	Lobster (claw)		<0·2						
	9	9	Lobster (body)	2·0–9·7	5·0						
	9	9	Lobster (tail)		<0·2						
	5	499	Cockles	<0·2–0·2	<0·2						
St David's Head to Anglesey	3	40	Mussels	0·2–0·3	0·3	17	17	2·90–86·0	19·9	<0·20–0·80	<0·20
	10	10	Lobster (body)	1·0–6·5	2·5						
	10	10	Lobster (claw)		<0·2						
	10	10	Lobster (tail)		<0·2						
	3	150	Portuguese oysters	0·5–0·6	0·5						
	1	10	Native oysters		0·2						

Table 5 (Cont'd)

Source	Shellfish other than crabs					Crabs					
	Number of analyses	Number of samples	Species	Range	Mean	Number of analyses	Number of samples	Brown Meat		White Meat	
								Range	Mean	Range	Mean
Anglesey to Solway	4	95	Mussels	<0·2–0·5	<0·35	15	15	0·4–5·5	2·1	<0·2–0·5	<0·3
	1	7	Oysters		0·3						
	4	78	Queens	<0·2–0·3	<0·2						
	36	62	Whelks	<0·2–2·6	<1·1						
	6	383	Brown shrimps	<0·2–0·5	<0·3						
	1	10	Native oysters		0·4						
Firth of Clyde	10	10	Scallop (whitemeat)	0·6–1·4	0·8						
	7	7	Scallop (gonad)	0·6–10·6	2·7						
	1	1	Winkles		0·2						
	8	100	Queens	0·2–0·4	0·3						
Firth of Forth	1	50	Winkles		0·21	61	67	0·5–8·2	2·7		0·02[(h)]

(a) Data from MAFF Fisheries Laboratory, Burnham and DAFS Marine Laboratory, Aberdeen.
(b) Includes samples taken in 1979.
(c) 10 samples.
(d) 43 samples.
(e) 72 samples.
(f) 156 samples.
(g) 114 samples.
(h) 1 sample.

Table 6: Cadmium intakes of high fish consuming groups[(a)]

Place	Number of individuals	Range of weekly intakes (μg)	Mean weekly intake (μg)
Southport	12	40–170	94
Blackpool	37	40–150	72
Morecambe	33	40–140	71
S W Cumbria	37	30–910	117
Plymouth	12	40–1310	344
Looe	17	60–1000	218
Brixham	26	40–1040	108
Total	174	40–1310	121[(b)]
Total diet samples	25[(c)]	90–190	126

(a) Results obtained by the Laboratory of the Government Chemist.
(b) Weighted average.
(c) Diets prepared from food purchased in 14 locations in the UK.

Table 7: Survey of Cadmium in vegetables grown on land not treated with sewage sludge (1976–1977)(a)

			Cadmium concentration (mg/kg)			
			Dry Weight		Fresh Weight	
Family	Vegetable(b)	Number of samples analysed	Mean(c)	Range	Mean(c)	Range
Brassica	Broccoli	6	<0·2	<0·1–0·5	<0·02	<0·01–0·03
	Brussels—Sprouts	9	<0·1	<0·1–0·1	<0·01	<0·01–0·02
	—Tops	11	<0·2	<0·1–0·6	<0·03	<0·01–0·08
	Cabbage	22	<0·1	<0·1–0·3	<0·01	<0·01–0·03
	Cauliflower	11	<0·2	<0·1–0·3	<0·02	<0·01–0·03
	Kale	12	<0·3	<0·1–0·7	<0·04	<0·01–0·10
	Swede	8	<0·3	<0·1–0·9	<0·03	<0·01–0·06
Umbelliferae	Carrots	13	<0·5	<0·1–0·9	<0·05	<0·01–0·11
	Celery	9	0·6	0·2–1·5	0·04	0·01–0·09
	Parsley	6	<0·2	<0·1–0·4	0·03	0·02–0·06
	Parsnips	10	0·4	0·1–1·9	<0·08	<0·01–0·37
Liliaceae	Leeks	14	<0·4	<0·1–1·8	<0·04	<0·01–0·16
	Onions	9	<0·2	<0·1–0·4	<0·02	<0·01–0·04
Compositae	Lettuce	17	0·8	0·1–2·4	<0·06	<0·01–0·39
Chenopodiaceae	Beetroot	15	<0·2	<0·1–0·5	<0·03	<0·01–0·06
Basidiomycetes	Mushrooms	4	0·8	0·2–1·4	0·06	0·02–0·08
Gramineae	Barley	21	<0·03	<0·01–0·06	<0·03	<0·01–0·05
	Oats	10	0·06	0·01–0·20	<0·05	<0·01–0·18
	Wheat	20	<0·05	<0·01–0·11	<0·05	<0·01–0·09
Leguminoseae	Broad beans	11	<0·1	<0·1–0·1	<0·02	<0·01–0·04
	French beans	11	<0·2	<0·1–0·5	<0·01	<0·01–0·03
	Runner beans	9	<0·1	<0·1–0·1	<0·01	<0·01–0·01
	Peas	23	<0·1	<0·05–0·40	<0·03	<0·01–0·06
Solanaceae	Potatoes	20	<0·1	<0·05–0·35	<0·03	<0·01–0·06
	Tomatoes	22	0·2	0·1–0·5	<0·01	<0·01–0·04
Cucurbitaceae	Cucumber	11	0·2	0·1–0·3	<0·01	<0·01–0·03

(a) Data from MAFF Food Science Laboratory.
(b) All vegetables were prepared as for cooking or direct consumption.
(c) Calculated by assuming that results below the limit of determination were at the limit.

Table 8: Cadmium in vegetables grown on experimental plots near to Heathrow[(a)]

Vegetable	Cadmium concentration (mg/kg fresh weight)	
	Plot treated with sludge over many years	Untreated plot
Lettuce	1·79, 0·14	0·06, 0·02
Carrots (roots)	0·69, 0·23	0·03, 0·06
Parsnips (roots)	1·47, 0·43	0·07, 0·05
Red beet (roots)	0·65, 0·36	0·07, 0·03
Leeks	0·71, 0·45	0·03, 0·02
Cabbage	0·16, 0·18	0·01, 0·01
Runner beans (pods)	0·03, 0·03	0·01
Total soil cadmium[(b)]	11	0·3
Soil pH	6·4	6·6
Soil organic matter	5·0%	2·2%

(a) Some of the results are taken from a paper by S. J. Richardson in, *Inorganic Pollution and Agriculture,* MAFF 1980.

(b) Total soil cadmium determined by HNO_3–$HClO_3$ digestion using standard ADAS procedure.

Table 9: Cadmium in vegetables grown in Heathrow market gardens (1978–1979)(a)

Vegetable	Total soil cadmium concentration (mg/kg)(b)	Cadmium concentration in vegetables (mg/kg fresh weight)		
		No. analysed	Range	Mean
Beetroot	<1	–		
	1–5	3	0·14–0·26	0·19
	5–10	1		0·14
	>10	1		0·42
Broccoli	<1	1		0·02
	1–5	–		
	5–10	3	0·05–0·18	0·10
	>10	–		
Cabbage	<1	5	<0·01–0·02	<0·01
	1–5	21	<0·01–0·03	<0·02
	5–10	3	0·03–0·06	0·05
	>10	6	0·03–0·08	0·06
Carrots	<1	–		
	1–5	–		
	5–10	1		0·11
	>10	1		0·19
Cauliflower	<1	–		
	1–5	4	<0·01–0·06	<0·03
	5–10	–		
	>10	1		0·08
Celeriac	<1	–		
	1–5	1		0·14
	5–10	–		
	>10	–		
Celery	<1	–		
	1–5	1		0·12
	5–10	2	0·25–0·26	0·25
	>10	–		
Courgettes	<1	–		
	1–5	–		
	5–10	1		0·02
	>10	1		0·02
Kale	<1	–		
	1–5	–		
	5–10	–		
	>10	2	0·10–0·22	0·16
Leeks	<1	3	0·01–0·06	0·03
	1–5	17	<0·01–0·17	<0·07
	5–10	4	0·14–0·30	0·20
	>10	–		

Table 9 (Cont'd)

Vegetable	Total soil cadmium concentration (mg/kg)(b)	Cadmium concentration in vegetables (mg/kg fresh weight)		
		No. analysed	Range	Mean
Lettuce	<1	2	0·04–0·07	0·06
	1–5	18	0·01–0·24	0·08
	5–10	12	0·03–0·35	0·17
	>10	18	0·15–0·59	0·31
Marrow	<1	1		0·01
	1–5	1		0·02
	5–10	1		0·02
	>10	1		0·03
Mint	<1	–		
	1–5	2	0·04–0·05	0·05
	5–10	–		
	>10	–		
Parsley	<1	–		
	1–5	2	0·10–0·15	0·13
	5–10	1		0·13
	>10	1		0·18
Parsnips	<1	–		
	1–5	3	0·10–0·13	0·12
	5–10	–		
	>10	–		
Potatoes	<1	–		
	1–5	2	0·06–0·17	0·11
	5–10	3	0·12–0·21	0·17
	>10	2	0·07–0·17	0·12
Radish	<1	2		0·02
	1–5	2	0·07–0·24	0·16
	5–10	1		0·11
	>10	–		
Spinach	<1	–		
	1–5	5	0·11–0·52	0·29
	5–10	3	0·37–1·26	0·74
	>10	3	0·41–1·07	0·68
Spinach beet	<1	–		
	1–5	–		
	5–10	1		0·50
	>10	–		
Spring greens	<1	1		0·02
	1–5	2	<0·01–0·15	<0·07
	5–10	1		0·07
	>10	6	0·01–0·16	0·10

Table 9 (Cont'd)

Vegetable	Total soil cadmium concentration (mg/kg)[(b)]	Cadmium concentration in vegetables (mg/kg fresh weight)		
		No. analysed	Range	Mean
Spring onions	<1	2	0·01–0·02	0·01
	1–5	7	0·02–0·37	0·14
	5–10	5	0·04–0·44	0·19
	>10	1		0·46
Sweet corn	<1	1		0·18
	1–5	3	0·06–0·19	0·11
	5–10	–		
	>10	3	0·46–1·23	0·73
Turnips	<1	–		
	1–5	3	0·02–0·06	0·04
	5–10	1		0·18
	>10	–		

(a) Analyses by MAFF Food Science Laboratory.
(b) Total soil cadmium determined by HNO_3–$HClO_3$ digestion using standard ADAS procedure.

Table 10: Cadmium in fruit and vegetables grown at Shipham (mg/kg fresh weight)[a]

Vegetable	Samples	Mean	Median	Range	% in each of the following ranges: Less than or equal to 0·05	Between 0·05 and 0·1	Between 0·1 and 0·2	Between 0·2 and 0·5	Greater than 0·5
Artichoke	6	0·15	0·12	0·07–0·29	0	50	17	33	0
Beans—Broad	25	0·08	0·07	0·01–0·20	28	52	16	4	0
—French	12	0·02	0·02	0·01–0·04	100	0	0	0	0
—Runner	60	0·03	0·02	0·002–0·30	97	2	0	2	0
Beetroot	30	0·36	0·35	0·04–1·14	3	3	17	60	17
Broccoli	4	0·39	0·13	0·08–1·23	0	50	25	0	25
Brussels—Sprout	46	0·17	0·14	0·02–0·79	4	22	43	28	2
—Top	1	0·24		–					
Cabbage	66	0·61	0·25	0·02–8·24	6	17	18	26	33
Cabbage—Red	5	0·54	0·38	0·02–1·70	20	0	0	60	20
Carrots	75	0·34	0·31	0·03–1·58	3	4	17	64	12
Cauliflower	1	0·02		–					
Celeriac	1	0·54		–					
Celery	2	1·0	1·0	0·40–1·68	0	0	0	50	50
Chard	1	0·55		–					
Chicory	2	1·5	1·5	1·5–1·6	0	0	0	0	100
Chive	7	1·0	0·49	0·1–2·4	0	0	29	29	43
Courgette	13	0·02	0·02	0·01–0·04	100	0	0	0	0
Cucumber	2	0·03	0·03	0·02–0·04	100	0	0	0	0
Gherkin	1	0·03		–					
Kale	19	0·75	0·35	0·08–1·23	0	11	21	26	42
Kohlrabi	4	0·10	0·08	0·04–0·17	25	50	25	0	0
Leeks	52	0·39	0·33	0·06–1·77	0	8	15	50	27
Lemon Balm	5	0·10	0·07	0·05–0·18	40	20	40	0	0
Lettuce	79	0·68	0·51	0·03–2·90	5	3	8	30	54
Marrow	6	0·03	0·02	0·01–0·13	83	0	17	0	0
Mint	27	0·14	0·10	0·02–0·4	26	26	19	30	0

Table 10 (Cont'd)

Vegetable	Samples	Mean	Median	Range	% in each of the following ranges				
					Less than or equal to 0·05	Between 0·05 and 0·1	Between 0·1 and 0·2	Between 0·2 and 0·5	Greater than 0·5
Mushroom	1	3·5		–					
Onion	33	0·22	0·19	0·03–0·44	3	12	36	48	0
Onion—Spring	9	0·22	0·20	0·07–0·36	0	11	33	55	0
Parsley	15	0·41	0·34	0·15–1·02	0	0	20	53	27
Parsnip	41	0·30	0·25	0·09–0·68	0	5	29	51	15
Pea	25	0·06	0·05	0·01–0·18	48	40	12	0	0
Pepper—Green	2	0·05	0·05	0·03–0·06	50	50	0	0	0
Potato	62	0·13	0·13	0·03–0·30	15	21	45	19	0
Radish	1	0·28		–					
Rosemary	1	0·42		–					
Sage	4	0·19	0·22	0·03–0·31	25	0	25	50	0
Salsify	1	0·26		–					
Sea Kale	1	0·42		–					
Shallot	4	0·20	0·20	0·18–0·23	0	0	50	50	0
Spinach	21	0·79	0·58	0·19–1·70	0	0	5	43	52
Spring Greens	6	0·55	0·31	0·07–1·80	0	17	33	17	33
Swede	9	0·13	0·11	0·06–0·28	0	44	33	22	0
Sweet Corn	1	0·34		–					
Thyme	5	1·6	1·32	0·15–3·20	0	0	20	0	80
Tomatoes	11	0·06	0·05	0·02–0·12	55	27	18	0	0
Turnips	14	0·23	0·20	0·09–0·52	0	7	43	43	7
Blackberry	1	0·08		–					
Blackcurrants	12	0·04	0·02	0·01–0·15	83	8	8	0	0
Gooseberry	16	0·03	0·02	0·01–0·09	89	11	0	0	0
Loganberry	4	0·12	0·10	0·02–0·24	50	0	25	25	0
Raspberry	15	0·10	0·07	0·01–0·39	40	27	20	13	0
Redcurrant	2	0·17	0·17	0·06–0·28	0	50	0	50	0
Rhubarb	71	0·42	0·35	0·04–1·20	3	10	18	35	34
Strawberry	1	0·01		–					

(a) Analyses by Somerset County Analyst's Department, and MAFF Food Science Laboratory.

Table 11: Comparison between cadmium in vegetables from Shipham and from Heathrow with cadmium in vegetables grown in non-contaminated soils

Vegetable	From Non-contaminated soils (Table 7)		From Heathrow (Table 9)		From Shipham (Table 10)	
	No. of samples	Mean cadmium concentration (range) (mg/kg)	No. of samples	Mean cadmium concentration (range) (mg/kg)	No. of samples	Mean cadmium concentration (range) (mg/kg)
Beans—Broad	11	<0·02 (<0·01–0·04)			25	0·08 (0·01–0·20)
—French	11	<0·01 (<0·01–0·03)			12	0·02 (0·01–0·04)
—Runner	9	<0·01 (<0·01–0·01)			61	0·03 (0·002–0·30)
Beetroot	15	<0·03 (<0·01–0·06)	5	0·23 (0·14–0·42)	30	0·36 (0·04–1·14)
Broccoli	6	<0·02 (<0·01–0·03)	4	0·08 (0·02–0·18)	4	0·39 (0·08–1·23)
Brussels—Sprouts	9	<0·01 (<0·01–0·02)			46	0·17 (0·02–0·79)
—Tops	11	<0·03 (<0·01–0·08)			1	0·24
Cabbage	22	<0·01 (<0·01–0·03)	35	<0·03 (<0·01–0·08)	68	0·61 (0·02–8·24)
Carrots	13	<0·05 (<0·01–0·11)	2	0·15 (0·11–0·19)	77	0·34 (0·03–1·58)
Cauliflower	11	<0·02 (<0·01–0·03)	5	<0·04 (<0·01–0·08)	1	0·02
Celery	9	0·04 (0·01–0·09)	3	0·21 (0·12–0·26)	2	1·04 (0·40–1·68)
Cucumber	11	<0·01 (<0·01–0·03)			2	0·03 (0·02–0·04)
Kale	12	<0·04 (<0·01–0·10)	2	0·16 (0·10–0·22)	19	0·75 (0·08–1·23)
Leeks	14	<0·04 (<0·01–0·16)	24	<0·09 (<0·01–0·30)	52	0·39 (0·06–1·77)
Lettuce	17	<0·06 (<0·01–0·39)	50	0·18 (0·01–0·59)	79	0·68 (0·03–2·90)
Mushrooms	4	0·06 (0·02–0·08)			1	3·5
Onions	9	<0·02 (<0·01–0·04)			33	0·22 (0·03–0·44)
Parsley	6	0·03 (0·02–0·06)	4	0·14 (0·10–0·18)	15	0·41 (0·15–1·02)
Parsnips	10	<0·08 (<0·01–0·37)	3	0·12 (0·10–0·13)	42	0·30 (0·09–0·68)
Peas	23	<0·03 (<0·01–0·06)			25	0·06 (0·01–0·18)
Potatoes	20	<0·03 (<0·01–0·06)	7	0·14 (0·06–0·21)	64	0·13 (0·03–0·30)
Swedes	8	<0·03 (<0·01–0·06)			9	0·13 (0·06–0·28)
Tomatoes	22	<0·01 (<0·01–0·04)			14	0·06 (0·02–0·12)

SPECIES OF FISH AND SHELLFISH SAMPLED

Common name	Generic name
Marine fish	
Anchovy	*Engraulis encrasicolus*
Bass	*Dicentrarchus labrax*
Brill	*Scophthalmus rhombus*
Cod	*Gadus morhua*
Coley	*Pollachius virens*
Dab	*Limanda limanda*
Dogfish	*Galeorhinus galeus*
Dover Sole	*Solea solea*
Five Beard Rockling	*Ciliata mustela*
Flounder	*Platichthys flesus*
Grey Mullet	*Crenimugil labrosus*
Gurnard (Red)	*Aspitrigla cuculus*
Haddock	*Melanogrammus aeglefinus*
Hake	*Merluccius merluccius*
Herring	*Clupea harengus*
Lemon Sole	*Microstomus kitt*
Lumpfish	*Cyclopterus lumpus*
Mackerel	*Scomber scombrus*
Megrim	*Lepidorhombus whiffiagonis*
Monkfish (Angler Fish)	*Lophius piscatorius*
Octopus	*Octopus vulgaris*
Pilchard	*Sardina pilchardus*
Plaice	*Pleuronectes platessa*
Pollack	*Pollachius pollachius*
Pouting	*Trisopterus luscus*
Queenfish	*Seriphus politus*
Rockfish, Rock Salmon, Catfish	*Anarhichas lupus*
Sardine	*Sardina pilchardus*
Skate	*Raja spp.*
Sprat	*Sprattus sprattus*
Tuna (Tunny Fish) for example	*Euthynnus pelamis* and *Thunnus alalunga*
Whiting	*Merlangus merlangus*
Witch	*Glyptocephalus cynoglossus*
Shellfish	
(Molluscs)	
Clam	*Mercenaria mercenaria*
Cockle	*Cardium edulis*
Mussel	*Mytilus edulis*
Oyster	*Ostrea edulis*
Queen Scallop	*Chlamys opercularis*
Scallop	*Pecten maximus*

Common name	Generic name
Winkle	*Littorina littorea*
Whelk	*Buccinum undatum*
(Crustaceans)	
Brown Shrimp	*Crangon crangon*
Crab	*Cancer paguris*
Japanese Prawn	*Pandalus japonicus*
Lobster	*Homarus vulgaris*
Norway Lobster or Dublin Bay Prawn	*Nephrops norvegicus*
Pink Shrimp	*Pandalus montagui*
Prawn	*Palaemon serratus*
Freshwater fish	
Bream	*Abramis brama*
Brown Trout	*Salmo trutta* which has spent all of its life in fresh water
Carp	*Cyprinus carpio*
Dace	*Leuciscus leuciscus*
Eel	*Anguilla anguilla*
Perch	*Perca fluviatilis*
Pollan (freshwater herring)	*Coregonus pollan*
Rainbow Trout	*Salmo gardinerii*
Rudd	*Leuciscus erythrophthalmus*
Salmon	*Salmo salar*
Sea Trout	*Salmo trutta*

APPENDIX III

MEMBERSHIP OF THE WORKING PARTY ON THE MONITORING OF FOODSTUFFS FOR HEAVY METALS

A W Hubbard (*Chairman* 1973–1980)	Ministry of Agriculture, Fisheries and Food
Dr P J Bunyan (*Chairman* 1980–)	Ministry of Agriculture, Fisheries and Food
T J Coomes	Ministry of Agriculture, Fisheries and Food
R F Clements	Department of Health and Social Security
Dr W H Evans	Laboratory of the Government Chemist, Department of Industry
Dr G I Forbes	Scottish Home and Health Department
Dr J L Facer	Department of Health and Social Security
R E J Goodman	Ministry of Agriculture, Fisheries and Food
K G Gostick	Ministry of Agriculture, Fisheries and Food
A V Holden	Freshwater Fisheries Laboratory, Department of Agriculture and Fisheries for Scotland
D F Lee	Harpenden Laboratory, Ministry of Agriculture, Fisheries and Food
Miss G Lewis	Central Veterinary Laboratory, Ministry of Agriculture, Fisheries and Food
Dr D J McWeeny	Food Science Laboratory, Ministry of Agriculture, Fisheries and Food
Miss H J Morey	Ministry of Agriculture, Fisheries and Food
Dr J E Portmann	Fisheries Laboratory, Ministry of Agriculture, Fisheries and Food
Dr L J Powell	Welsh Office
A H Strickland	Harpenden Laboratory, Ministry of Agriculture, Fisheries and Food
J F A Thomas	Department of the Environment
Professor J R Todd	Department of Agriculture for Northern Ireland
Dr G Topping	Marine Laboratory (Aberdeen), Department of Agriculture and Fisheries for Scotland
Dr J F C Tyler	Laboratory of the Government Chemist, Department of Industry

Secretariat

Scientific:

Dr J C Sherlock	Ministry of Agriculture, Fisheries and Food
Miss G A Smart	Ministry of Agriculture, Fisheries and Food
C B Walters	Ministry of Agriculture, Fisheries and Food

Administrative:

A Flower	Ministry of Agriculture, Fisheries and Food

APPENDIX IV

REPORT OF THE COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

SURVEY OF CADMIUM IN FOOD

INTRODUCTION

We were requested by the Steering Group on Food Surveillance to consider the report on the Survey of Cadmium in Food, First Supplementary Report, compiled by the Working Party on the Monitoring of Foodstuffs for Heavy Metals (WPMFHM) and in the context of the report of the investigations into the pollution by cadmium at Shipham to assess hazards to health that could arise from cadmium in food and to review present information on the toxicity of cadmium. We reported previously on Cadmium in food in 1973[1].

Our views on the Survey of Cadmium in Food, First Supplementary Report, and an assessment of possible health hazards from the quantities of cadmium in the United Kingdom diet are presented in Section A.

A review of cadmium pharmacokinetics and toxicology (with special emphasis on chronic toxicity) is presented in Section B.

Section A: Cadmium in food in the United Kingdom:
1. Exposure to cadmium
2. Dietary cadmium
3. Permitted cadmium levels in food
4. Summary and conclusions

Section B: Toxicology of cadmium:
1. Absorption of cadmium
2. Transport and distribution of cadmium
3. Body burden of cadmium
4. Excretion of cadmium
5. Accumulation of cadmium
6. Toxic effects of cadmium in man
7. Nephrotoxicity of cadmium
8. Assessment of a safe level of long-term cadmium intake

CADMIUM IN FOOD

A1. Exposure to Cadmium

Cadmium is absorbed by ingestion and inhalation. Ingested cadmium is the major source of cadmium intake for the general public, with food providing by far the greater part and water contributing only a little. Tobacco can be an important source.

The average intake from drinking water is assessed at 1–2 μg/day[2].

The general air contributes very small amounts to the total cadmium intake. In urban areas, the long-term average intake is unlikely to exceed 0·4 μg/day[2].

Cadmium intake can be increased by raised amounts in food, especially in shellfish, water and occupational exposure to cadmium fumes or dusts. Tobacco contains cadmium and smoking, particularly of unfiltered cigarettes, can be as important a source of cadmium as the diet.

A2. Dietary Cadmium

2.1 *Monitoring surveys*

Information collated from surveys carried out over the years 1973–1981 by the Ministry of Agriculture, Fisheries and Food (MAFF), the Laboratory of the Government Chemist (LGC) and the Department of Agriculture and Fisheries for Scotland (DAFS) published in the 12th report of the WPMFHM entitled, *Survey of Cadmium in Food: First Supplementary Report,* allows an assessment of the current dietary intake of cadmium in the general population of the United Kingdom.

These surveys comprised measurement of the average daily intake of cadmium using the methodology of the Total Diet Study[3] and the cadmium content of a very wide range of foodstuffs. Data were accumulated also on the cadmium content of marine and freshwater fish and shellfish, and these were complemented by duplicate diet studies among high fish consumers in five coastal areas. Further data derived from duplicate diet studies among populations living on soil contaminated with cadmium.

The FAO/WHO provisional tolerable weekly intake (PTWI) for cadmium is 400/500 μg and the margin of safety between this and actual intakes in the United Kingdom is not great. We consider therefore that similar surveys should continue. However, priority should be given to studies of foodstuffs high in cadmium. Further *ad hoc* studies will be required to investigate any new sources of contamination by cadmium that are found and long-term surveillance of affected populations is recommended.

We note that there is insufficient information to enable advice to be given on a tolerable intake of cadmium for children, and consider that any research opportunities that may present in this field should be followed up.

2.2 *Dietary Cadmium intake*

In the United Kingdom food is normally the major source of cadmium and in their report MAFF estimate that the mean daily intake is less than 20 μg, corresponding to a mean weekly intake of less than 140 μg. The Total Diet Studies indicate that under normal circumstances the cereal group of foods is the largest single contributor of cadmium, but other studies have demonstrated that in unusual circumstances vegetables, and rather more frequently shellfish, may become the major contributors.

The Total Diet Studies indicate that the mean intake of cadmium for people living in the United Kingdom is below the FAO/WHO PTWI and these data indicate that the range of intakes is fairly restricted (120 sample diets showed a mean intake of 131 μg/week, and a standard deviation of 26 μg/week). We note that there are rather small margins between the level of cadmium intake that might cause toxic manifestations in sensitive individuals, the FAO/WHO PTWI, and the actual intakes reported for the United Kingdom.

2.3 *Cadmium in shellfish*

A consideration of body intakes of metals purely in terms of the mass ingested may neglect differences in the quantities absorbed from different foods between which the availability of a metal may differ. In the case of cadmium there is evidence[4, 5] that the solubility of the metal contained in shellfish is low and we have noted the recent work by Newton[6] which found that the mean retention within the human body of cadmium ingested in crabmeat was approximately one-half of the retention that has been reported for other foods. We consider that this finding justifies a modification of the concern that would otherwise extend to those with the most extreme intakes of shellfish.

2.4 *Cadmium in vegetables*

A study in the Heathrow area, where vegetables are grown on land contaminated from sewage sludge applied as a fertiliser, showed that although some parts of the area were heavily contaminated, and that this led to raised cadmium levels in vegetables grown there, the cadmium levels in the diets were raised by less than 50 per cent (Total Diet Study 17 μg/kg, Heathrow 23 μg/kg) and further studies indicated that the intakes at Heathrow were unlikely to exceed the PTWI for cadmium.

We note the extensive dietary studies that have been completed at Shipham, where parts of the village are on soil very heavily contaminated from mining activities in the past. The studies indicate that the dietary cadmium levels are nearly double those found in the national diet; it appears probable that about 4 per cent of the population studied may consistently exceed somewhat the PTWI. We note that the problem would be much reduced if a small range of vegetables was no longer grown in the village, and that cadmium intake can be held at safe levels by the careful washing of vegetables. Such precautions cannot, however, be wholly reliable and we recommend that there should be further monitoring, dietary and medical, at Shipham.

A3. Permitted Cadmium levels in food

There are no regulations relating to cadmium in food sold in the United Kingdom. The levels of cadmium reported by MAFF do not indicate that there is a strong toxicological basis for the setting of standards for cadmium in food.

A4. Summary and conclusions

We consider that while the average dietary intake of cadmium does not in itself give cause for concern, it nevertheless represents a considerable proportion of the accepted tolerable intake and merits continued monitoring, especially of foodstuffs known to accumulate cadmium to excess.

Studies of heavily exposed populations in the United Kingdom have shown that a very small proportion of such people may exceed the accepted tolerable intake for cadmium, though only exceptionally is this intake grossly exceeded. A high consumption of some shellfish could lead in theory to considerably raised intakes of cadmium but experience suggests that this is a very rare event. A high dietary intake combined with heavy smoking over a lifetime could in theory lead to a toxic body burden of cadmium, but the indications are that this will be a most rare phenomenon in the United Kingdom and such cases are not known to us.

Theoretical considerations based on the data that are available to the Committee do not indicate that the total cadmium intakes from food, drinking water and environmental air in the United Kingdom pose any significant hazard to health.

We note the paucity of information, dietary and toxicological, that relates to children. We recommend that:

1. Monitoring of food for cadmium should continue, but should, in future, concentrate upon foodstuffs of special concern.
2. Dietary studies should concentrate on specially exposed groups and children.
3. There should be further studies, whenever an indication is found, to determine the availability to humans of cadmium in specific food species.

TOXICOLOGY OF CADMIUM

B1. Absorption of Cadmium

For non-occupationally exposed populations the significant routes of absorption are the gastro-intestinal tract and by the lungs. For humans absorption through the skin does not appear important; animal experiments indicate that less than 2 per cent of a cadmium chloride solution may be absorbed after 5 hours skin contact[7].

1.1. *Absorption via the intestinal tract*

Experiments have shown that in animals the gastro-intestinal absorption of cadmium varies with the compound given and with the animal species; a range of 0·5–8 per cent has been quoted by Friberg *et al.*[8].

In human studies, cadmium has been found to have an unusually long dwell-time in the gut, which makes balance studies, including those utilising radioactive isotopes of cadmium, unusually difficult. Rahola *et al.*[9] (in five humans, aged 19–50 years) found absorption rates in the range 4·7–7 per cent, which agree with calculations made by Friberg[8]. The Commission of the European Communities (CEC) concluded that the average oral absorption in adult man is 5 per cent[10].

Animal experiments indicate that nutritional status may affect cadmium absorption, which may be increased by deficiencies of calcium, iron and protein. Human data are not available; but it is noted that in the general environment cadmium toxicity in its most extreme form (Itai-Itai disease) has been reported only in populations whose nutritional status was poor.

There are two reports[4, 5] that (*in vitro*) the cadmium bound to lobster and crab protein is less soluble and possibly less available than that in other foods. Newton[6] has investigated the availability of cadmium from brown crabmeat using a radioactive isotope and whole body counting. In seven subjects, after three months, the retention was 1·8 per cent (geometric mean) or 2·6 per cent (arithmetic mean).

1.2 *Absorption by inhalation*

The cadmium content of the air in the general environment in the United Kingdom, with the exception of one reported hotspot, rarely exceeds 0·02 to 0·03 μg/m³ even in industrial areas[2]. Even if all the cadmium in such inhaled air were deposited in the alveoli and absorbed, it would only amount to 0·30–0·45 μg/day which is not in itself a significant dose. There are few data from which the absorption can be deduced, but by taking into account the range of particle size and calculations from data published by Lewis[11] the CEC concluded that about 25 per cent of the cadmium inhaled from environmental air may be absorbed[10].

Cigarette smoking can provide a very important increment to cadmium levels in air. Lewis[11] reported that smoking one packet of cigarettes per day for 40 years more than doubled the mean combined cadmium levels of kidneys, lungs and liver (from 6·8 to 14 mg). Data on the deposition and absorption of cadmium in tobacco smoke are not available. However, it is reported that the particle size is <0·3 μ and the CEC[10] concluded that overall absorption might be 32 per cent. However, this calculation ignored excretion and assumed that such cadmium has a biological half-life similar to that of cadmium acquired by other routes. The truth of this will not be known until cadmium balance studies in smokers are reported.

B2. Transport and distribution of Cadmium

2.1 *Transport of Cadmium*

Information on the transport of cadmium from the sites of absorption to the tissues in which it is deposited is found exclusively in reports of animal experiments.

In animals, absorbed cadmium enters the plasma from which it is cleared rapidly; Nordberg found the erythrocyte concentration to be 100 times that in plasma after a single injection[12]. Similar erythrocyte plasma ratios were found after repeated injections by Nordberg *et al.*[13], but the dosages were too large for any useful extrapolation to humans, consisting of 0·25 mg Cd on 5 days a week for 6 months (in mice). Webb[14] considers that it is probable that 30 per cent or more of the cadmium in erythrocytes is thionein-bound, and it is likely that thionein is not released into the plasma. In plasma the cadmium is thought to be bound to high molecular weight (70 000) protein.

Experiments in which isolated metallothioneins have been injected indicate that these are rapidly taken up by the kidney. The permeability of the glomeruli to high molecular weight protein bound cadmium has not been elucidated. Renal uptake of cadmium may involve luminal and peritubular cells.

2.2 *Distribution of Cadmium*

Data on tissue concentrations of cadmium in humans have been reported by Friberg[8] and by Sumino[16]. The highest concentrations are found in the kidney (cortex: whole kidney 1·5:1) followed by the liver. The liver/kidney ratios may change when nephrotoxicity occurs (see section B3). In animals, high dosages of cadmium lead first to accumulation in the liver, followed by a redistribution to other tissues that is comparable to human findings.

B3. Body burden of Cadmium

It used to be thought that fetal tissues contained no cadmium, (but recently Arena has demonstrated levels up to 50 μg/kg[49]) and that after birth the body burden gradually increases to the age of 50 years. Friberg's[8] review of the data suggests that the range of accumulation to this age may be 10–80 mg (whole body burden) in people not excessively exposed. Factors that influence an individual body burden include age, smoking habits, diet and occupation. In a single geographical area great individual differences were reported by Tsuchiya[17]; there are indications that the distribution of values is log-normal[18].

There is also general agreement that in cases of long-term general environmental exposure the critical organ is the kidney, which contains about one-third of the body burden[8, 10]. Data available on the concentrations found in renal cortex relate quite well to the range of whole body burdens quoted by Friberg. In renal cortex from subjects aged 40–59 years, Anke and Schneider[19] found arithmetic mean values of 26·5 (men) and 13·5 (women) μg/g wet weight; this relates to the German Democratic Republic. Scandinavian data are similar; a combination of male and female data yielding averages in the range 23–29 μg/g[18, 20]. The USA and Japan report rather higher levels.

Kjellstrom[21] has provided confirmation of these results in the course of a study, one of whose aims was to harmonise analytical performance in different countries. He found average burdens at age 45 years of 21 mg (Tokyo), 9 mg (Dallas) and 6 mg (Stockholm). All these levels relate well to the extensive data cited and reviewed by CEC[10].

After the age of 50 years, the renal cortical concentration of cadmium is known to decrease. The reason for this has not been ascertained, but after considering the possibilities (including changes in the metabolism with age, and demographic considerations) the CEC[10] concluded that the most plausible explanation was the increased exposure of populations over the last 50 years, and especially the last 25 years. (It was estimated in 1975 that 70 per cent of total world production had occurred in the previous 20 years.) If this is the case, the age at which the peak body burden is observed will rise; so far there are no firm indications that this is happening.

Approximately one-third of the body burden in normal individuals is found in the kidneys, and one-sixth in the liver. When a critical concentration is reached in kidney, the liver/kidney ratio is disturbed; concentrations in liver continue to rise but nephrotoxicity results in a fall, which may be very considerable in kidney levels, due to excretion. Thus renal concentrations after nephrotoxicity has developed no longer provide reliable data from which to judge body burden or critical concentration. It is debatable whether these considerations were adequately allowed for when the World Health Organisation's Task Group assessed the probable renal cortical critical concentration at 200 μg/g wet weight[22, 23, 24], and it is arguable that this figure may be rather low as it is known that the data considered included some derived from occupationally exposed persons with nephrotoxicity.

B4. Excretion of Cadmium

The principal route for excretion of absorbed cadmium is urinary. Reports differ widely in their assessment of urinary excretion and the data are not always related to such crucial factors as age, smoking habits or body burden. The data have been extensively reviewed and discussed by Friberg[8] and the CEC[10]. There are further difficulties in interpreting the data due to differences in the units of reporting (μg/l; μg/24 hr; μg/g creatinine) and to a general awareness of wide interlaboratory variations in analytical performance. However, it appears that in general there is a concensus that the mean urinary excretion in populations not excessively exposed to cadmium is in the range 0·5–2·0 μg/l; that it increases with age to level off after 40 years; that in occupationally exposed populations much higher excretion rates can occur without evidence of nephrotoxicity and that when this is evident several hundred micrograms of cadmium may be excreted daily.

Animal experiments indicate that cadmium can be excreted by the bile and gastro-intestinal tract, but that this is minimal[25] except when the cadmium is administered intravenously[26]. There are no data available on net gastro-intestinal excretion in humans, but it is believed to be very low and faecal cadmium levels have been used as an index of exposure[21].

Small amounts of cadmium are lost from hair, milk and the products of gestation.

B5. Accumulation of Cadmium

Animal experiments have been used in attempts to estimate the biological half-life of cadmium but have yielded disappointing results. It appears that there is wide variation between the species but that all are short relative to that believed to occur in humans[8].

Epidemiological studies have indicated a long biological half-life for cadmium. Mathematical models indicate that in the critical organ, the kidney, the half-life may be in the range 17–30 years[17, 18, 27]. Estimates of whole body half-time range from 10–30 years[8], possibly dependent on age[28].

Human experimental data are very few indeed. Five volunteers were fed a single dose of Cd^{115} and the results suggested a biological half-time between 130 days and infinity[29]. Newton[6] has reported a similar result. There are very great problems in studies using Cd^{115} due to the relatively short radiological half-time of the isotope.

Although the accumulation of cadmium reflects the balance between absorption and excretion, the rate of absorption may also be a determining factor. It is generally believed that excretion occurs in both rapid and slow phases. The former is assumed by Friberg[8] to be 10 per cent in chronic, low levels exposure and the latter largely determines the biological half-time. The rapid phase may considerably exceed 10 per cent in cases of occupational exposure, in the absence of nephrotoxicity[30].

B6. Toxic effects of Cadmium in man

6.1 *Acute exposure*

Consumption of foodstuffs contaminated by cadmium plated containers has caused severe gastro-intestinal disturbances. In some cases there was early death due to shock, in others death from renal failure and cardio-pulmonary depression occurred after 7–14 days[31]. Liver damage has been reported[32]. The emetic threshold has been estimated to be in the range 3–90 mg, the lethal dose to be 350–3500 mg[10].

Inhalation of excessive concentrations of cadmium fumes may cause severe respiratory symptoms, typically after a latent interval of several hours. Gastro-intestinal symptoms may also occur and in the most severe cases pulmonary oedema may develop.

6.2 *Chronic low-level exposure*

Data on the consequences of prolonged occupational exposure to cadmium are plentiful, but there are comparatively few data relating to environmental exposure. It is generally agreed that such exposure carries the potential risk of damage to the critical organ (kidney) but attempts to assess the risk rely on extrapolation from occupational experience; studies of the Itai Itai in Japan (see 6·4) are also informative but there other factors also operated (e.g. severe malnutrition) and other pollutants were present.

6.3 *Effects of Cadmium in humans*

Cadmium is one cause of the multiple proximal renal tubular defects that comprise the Fanconi syndrome. Characteristic features include:

- Excretion of low molecular weight proteins (including B_2 microglobulin and lysozyme)
- Glycosuria
- Generalised amino-aciduria
- Phosphaturia
- Uricosuria

Less frequently reported are:

- Bone disease
- Acidosis
- Potassium loss
- Sodium loss
- Calcium loss

Conditions which have been associated with cadmium exposure include:

(a) Cardiovascular Animal experiments have produced conflicting results, with effects in some strains of some species on exposure to cadmium, but not in others. Hypertension has not been reported as a feature of human exposure (occupational or environmental)[10], although tissue profiles for cadmium appear to be distorted in several diseases including hypertension.

(b) Cancer Only in the single instance of prostatic carcinoma is there any indication of a risk from malignancy and even in this case the numbers reported are small and no definite conclusion can be reached[33, 34]. The occurrence of sarcomas at the site of local injection of cadmium salts in rats is considered to constitute unreliable evidence of cancer risk to humans exposed to cadmium by the ingestion and/or inhalation routes. Similarly the occurrence of interstitial cell tumours of the testes as a late consequence of the exposure of rats to the administration of cadmium is considered to be a non-specific consequence of testicular atrophy. Substantial evidence of non-carcinogenicity at other sites reinforces this view.

(c) Mutagenicity The evidence in humans is conflicting; some studies note chromosome abnormalities in cadmium workers, but others do not. Experimental studies, both of chromosomes and using lethal dominant assay, have also been inconclusive.

(d) Teratogenicity In a comprehensive review of the data, Carmichael *et al.*[48] conclude that as there is evidence of a teratogenic effect in experimental animals it is reasonable to predict the possibility in humans. In animals there may be a threshold for acute exposure below which effects do not develop, and for humans this may be of minor importance, except perhaps for occupationally exposed pregnant women.

(e) Metabolic effects There is evidence from animal experiments that cadmium may distort hepatic and renal carbohydrate metabolism, possibly both by stimulating conversion of glycogen to glucose and by stimulating the activity of neoglucogenic enzymes. Slight anaemia is common. Minor abnormalities in liver function tests are reported after many years of occupational exposure.

(f) Miscellaneous effects Anosmia and yellow discolouration of teeth occurs in workers exposed to cadmium dust or fume. One study suggested an effect on the birthweight of babies born to women cadmium workers.

6.4 *Itai Itai Disease*

This condition has provided very nearly, if not quite all, the information that is available on cadmium as a definitive hazard in the general environment. The full syndrome is limited to multiparous, post menopausal women and consists of a painful osteomalacia with tenderness, skeletal deformity and liability to fracture; increased serum alkaline phosphatase activity and reduced serum phosphate; hypochromic anaemia; decreased ability to absorb fat; proteinuria, glycosuria and a variably raised cadmiumuria.

Events that were associated with the outbreak included a period of severe malnutrition during the war years; severe flooding in the Jintzu valley in 1945 and 1946 which mobilised tailings from a zinc/copper mine and led to contamination of agricultural areas, the rice crop and drinking water. A good agreement was found between levels of cadmium in soil and rice, and the incidence of Itai Itai disease. The majority of cases probably occurred in the decade following 1950. In 1967, 6717 subjects over 30 years of age were examined in the area; 50 cases were found, another 48 suspected and 136 showed urinary abnormalities. The incomplete syndrome, without bony abnormalities, occurred in men as well as women, and in other parts of Japan where cadmium pollution was identified. Although some Japanese authorities deny the link with cadmium and consider the aetiology to include malnutrition, lack of Vitamin D and exposure to multimetal pollution, general opinion in Japan appears to accept cadmium as the main factor and the Japanese authorities have designated 13 areas for surveillance on the criteria that drinking water exceeds 0·01 ppm and rice exceeds 0·4 ppm of cadmium.

B7. Nephrotoxicity of Cadmium

7.1 *Pathogenesis*

Chronic exposure to cadmium, if continued at a sufficient level for long enough may cause changes in renal function that include enzymuria, polyuria, aminoaciduria and glycosuria; changes in the handling of uric acid, calcium and phosphate; and proteinuria, which may be of high or low molecular weight or both. It was formerly held that low molecular weight proteinuria due to tubular damage was characteristic of the condition and occurred before any appearance of the larger urinary protein molecules which relate to glomerular disease. However, Lauwerys[35] and his co-workers have demonstrated that in occupational exposure, damage to the glomeruli and to the tubules occurs independently and without temporal relationship. Low molecular weight proteins include B_2 microglobulin, retinol-binding protein, ribonuclease, light chain immunoglobulin and lysozyme[36]. A large number of high molecular weight proteins have been demonstrated, including immunoglobin G, albumin, and Bence-Jones protein[37].

Cadmium accumulation in the kidney typically presents in two phases. The first phase is represented by 'silent' accumulation of cadmium, the second phase by functional renal changes indicative of proximal tubular damage. The phases change quite suddenly when a certain level of cadmium has accumulated and although it is possible that the very earliest stage (enzymuria) may be reversible the later stages (proteinuria) appear not to be. The amount of cadmium in the kidney at the time when the phases change is known as the 'critical concentration' and the intake of cadmium that has led up to this concentration is the 'critical dose'. Webb has reviewed the evidence that the nephrotoxic effects of cadmium are modified by metallothionein[46].

The mechanism of the phenomenon described has not been conclusively demonstrated. The hypothesis that has gained the widest acceptance is that associated with workers at the Karolinska Institute. They postulate that in the presence of normal glomerular filtration rate, an increased excretion of B_2 microglobulin indicates a failure of resorption by the proximal tubules[8]. This failure, they consider, comes about when the cadmium presented to the tubular cells exceeds the capacity of those cells to bind it in a non-toxic form; cadmium then damages the cells directly, and dis-placing zinc from certain enzymes (e.g. leucine amino peptidase)[38] further impairs the resorption and/or catobolism of proteins[39]. Glomerular damage, indicated by high molecular weight proteinuria, is considered to be due to a selective effect of cadmium on glomerular permability[35].

7.2 *Macroproteinuria*

This subject is poorly documented. In 1977 a major review sponsored by the CEC[10] found that reports of macroproteinuria as a feature of early cadmium toxicity came from only one laboratory, the one associated with Lauwerys, and the only report since then known to DHSS is from the same source[35]. While the occurrence of macroproteinuria is not in dispute, the quantities reported do not suggest that there is a primary effect on the glomerulus.

7.3 *B_2 Microglobulin*

Enquiries in the UK generally elicit the information that a 'normal' upper limit for urinary B_2 microglobulin may be about 60 μg/l, but for epidemiological purposes (as at Shipham) it has proved necessary to compare control populations with the exposed, due to lack of data on the range of values to be expected in any particular region of the country. A literature search has shown that some guidance is available although the quantity of data is not large and does not relate closely to the environmental problems in the UK. Three Swedish studies provide comparable conclusions (Table 1).

Table 1

Arithmetic mean	SD	95th percentile	Investigator
120 μg/24 hrs	40	200	Peterson[42]
75 μg/24 hrs	52	180	Evrin[43]
104 μg/l		290 μg/l	Kjellstrom[44]

To these may be added data reported from two unpolluted areas of Japan (Table 2).

Table 2

No.	Arithmetic mean (μg/g–cr)	Investigator
30	110	Tsuchiya[40]
40	140	

In all these studies, B_2 microglobulin was assayed by a radioimmune assay method.

7.4 *Dose-response B_2 microglobulin/Cadmium exposure*

Nearly all the data on the dose-response to cadmium relate to two groups: occupationally exposed persons and Japanese living in highly cadmium polluted areas. In both groups investigations have aimed at ascertaining the incidence of cases which have received a critical dose rather than at discovering dose-response relationships at sub-critical intakes of cadmium. Cellular damage is known to occur when the critical dose has been taken, but at lower intakes only functional changes occur. There is thus a difference in the nature of the phenomena observed at pre-critical and post-critical stages and extrapolation from high to lower levels of intake cannot be justified on the accepted hypothesis of cadmium nephropathy. To investigate the problem, Tsuchiya[40] has studied populations living in polluted and non-polluted areas of Japan. Statistical analysis indicates that B_2 microglobulin excretion is strongly linked to age, and less strongly to cadmium exposure; however, there are indications that B_2 microglobulin excretion may correlate to all levels of exposure.

It has recently been reported by Lauwerys[47] that B_2 microglobulin is unstable in acid urine, an 80 per cent loss occurring in 2 hours at pH 5. When urines with pH $<5{\cdot}6$ are omitted, B_2 microglobulin correlates very well (0·94) with retinal binding protein (RBP) which is stable in urine. Lauwerys recommends that RBP is a better urinary indicator of cadmium exposure than B_2 microglobulin. When comparing exposed populations with controls using B_2 microglobulin as an indicator, it may be wise to exclude urines more acidic than pH 5·6.

7.5 *Biological significance of Cadmium-related proteinuria*

(a) High molecular weight proteinuria This is very poorly documented. Such data as there are may indicate that the prognosis in such cases is poor.

(b) Low molecular weight proteinuria This condition is also poorly documented. It would seem that a raised excretion of B_2 microglobulin is not *per se* of much significance and such cases do not appear to progress to advanced degrees of renal failure. The appearance of a raised excretion of B_2 microglobulin can be of prognostic import in that most authors recognise that it usually precedes the other disturbances[10]. The significance of minor elevations in an individual is very poorly documented. Similarly the significance of the finding that of two populations one has a significantly higher B_2 microglobulin excretion than the other, both being broadly within the range considered normal, is quite unknown.

B8. Assessment of a safe level of long-term cadmium intake

Two methods have been devised for assessing the environmental hazard of cadmium to man. The first method is based upon observations made on humans and experimental animals; it suffers from the drawbacks that (a) few data are available, and these few relate to occupational exposure or to the Itai Itai incident (neither of which can be extrapolated with confidence to environmental conditions in UK), and (b) the data indicate that animals handle cadmium very differently from man, particularly in respect of the biological half-time (Section B5).

The second method is developed from the assumption that in humans there is a relationship between exposure and concentration in the target organ, and on certain assumptions (which can, however, be sustained) regarding the metabolism of cadmium by man. Both methods have been reviewed by the CEC[10].

In relation to oral exposure, the first method rests on data from epidemiological studies in Japan which indicated that the prevalence of kidney damage began to rise at a daily intake of about 200 μg of cadmium. The Japanese received this cadmium with rice, and in those circumstances an absorption co-efficient of 0·06 was estimated. Thus the threshold for toxicity is estimated by this method to be an absorbed dose of 12 μg/day. It was pointed out in Section B6 that the Japanese population at risk did not correspond to any UK group, being severely undernourished and exposed to other pollutants. It is known that cadmium reacts with other food components in a way that has not yet been evaluated. Calculations based only on such data must therefore be treated with caution. It is believed that the Japanese were exposed to negligible amounts of cadmium from the air or from smoking, but for brief periods drank a polluted water supply.

The second method was suggested by Friberg[8] and requires acceptance of several assumptions:

One-third of body burden is in the kidneys, and renal cortical concentration is 50 per cent above mean renal value.

Excretion is 10 per cent (rapid component) and 0·005 per cent (slow component).

Absorption is 4·5 per cent (gut) and 25 per cent (lungs).

The critical concentration in renal cortex is 200 μg/g (wet weight).

Friberg calculated that if all the cadmium is taken orally, the daily intake that would result in a critical renal concentration by the age of 50 years is 248 μg. This implies an absorption of 10 μg/day. The model also implies that if the cadmium all comes from the air, an air level of 2 μg/m^3 would be critical.

The conclusions to be drawn from this model can only be tentative. Much reliance has to be placed on the estimate of 200 μg/g as the critical renal cortical concentration, and although this level has received endorsement from WHO and other authorities[45], there are still reservations. For example, the data that were considered are relatively sparse, and some related to kidneys that had been damaged and may be supposed to have lost some of their cadmium (Section B3). Also, the Japanese data relate to a small number of cases that occurred in a very large exposed population and may reflect the experience of the most sensitive individuals. Thus, it seems that the human data may have indicated a value for the critical concentration that is below the mean. A recent study[45] that would support this view employed neutron activation measurements of an exposed population (82 smelter workers) and found critical values to be 300–400 μg/g for renal cortex.

The approach suggested by Friberg has been developed in recent years. The most recent study is by Kjellstrom and Nordberg[14] who have developed an eight compartment kinetic model. Their assumptions (most importantly their value for renal cortical critical concentration) are much the same as Friberg's, but their model indicates that the critical renal level would be reached by a daily cadmium intake with food of 440 μg (European) or 325 μg (Japanese).

The safety margins that obtain in relation to the United Kingdom dietary intake of cadmium are set out below.

	Critical intake (μg/day)	Actual intake (μg/day) for UK	Safety margin
Epidemiological studies	200	20	1:10·0
Friberg	248	20	1:12·4
Ellis	about 350	20	1:17·5
Kjellstrom (European)	440	20	1:22·0

The FAO/WHO PTWI is 400–500 μg/week. If the higher figure is compared with the critical intakes quoted, the safety margin ranges from 2·8–6·2.

There are other considerations to be taken into account when assessing safety. The effect of water may be discounted (Section A1) as it is very small. The same is the case for air in the general environment, even in close proximity to industry[2]. However, in 'hotspot' situations the CEC estimated that 25 μg/day might be deposited in the lungs, corresponding to an absorption of

16 μg, and as there is evidence that one such 'hotspot' has existed in the United Kingdom[2] the possibility that there may be others cannot be ruled out. Smoking can be an important source of cadmium; calculations from Lewis'[11] data indicating that for each pack of cigarettes smoked per day, 1 μg of cadmium is retained in the long term. Such retention from food (assuming a co-efficient of absorption of 0·05) would result from the ingestion of 20 μg of cadmium so that as a source cigarettes may equal or exceed food. In the case of shellfish, a study[6] has shown that the mean retention of cadmium is, at 2·6 per cent, about half the value assumed in the kinetic models.

There is no guidance available on what might be considered a safe intake of cadmium by children.

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APPENDIX V

REPORT OF THE FOOD ADDITIVES AND CONTAMINANTS COMMITTEE

1. After consideration of the report, *Survey of Cadmium in Food. First Supplementary Report,* at a meeting in July 1982 the Committee endorses the conclusions of the Working Party and the recommendations of the COT. The Committee recommends that industry find alternatives to cadmium and it also supports efforts to ensure that pollution of the environment by cadmium is reduced, especially where this pollution may affect the food supply. At present there are no recommended or statutory limits in the UK on cadmium in food. Taking into account the comments of the COT the Committee considers that the present evidence does not justify the imposition of statutory limits for cadmium in food. However, in support of its policy to reduce the cadmium concentration in food the Committee recommends that:

a) where there are controls on the cadmium content of food in other countries, enforcement authorities should have regard to these controls when examining food imported into the UK from these countries;

and

b) the Working Party should monitor the effects on the food supply and on the diet of efforts to reduce pollution of the environment with cadmium, and, where possible, should identify and eliminate the major sources and routes of contamination of the food supply with cadmium.

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